Human OCTN2/SLC22A5 Alexa Fluor® 647-conjugated Antibody

Monoclonal Mouse IgG_{2A} Clone # 893753

Catalog Number: FAB11004R

100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human OCTN2/SLC22A5 in ELISA.
Source	Monoclonal Mouse IgG_{2A} Clone # 893753
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	NS0 transfected with human SLC22A5 Accession # O76082
Conjugate	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 nm
Formulation	Supplied 0.2 mg/mL in a saline solution containing BSA and Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

Flow Cytometry	Titration recommended for optimal concentration with starting range of 0.1-1 µg/1 million cells. Sample used for this experiment was HEK293 Human Cell Line Transfected with Human OCTN2/SLC22A5 and eGFP

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Protect from light. Do not freeze.** • 12 months from date of receipt, 2 to 8 °C as supplied.

BACKGROUND

Polyspecific organic cation transporters in the liver, kidney, intestine, and other organs are critical for the elimination of many endogenous small organic cations as well as a wide array of drugs and environmental toxins. SLC22A5 is an integral plasma membrane protein which functions as an organic cation transporter and as a sodium-dependent high affinity carnitine transporter. Mutations in SLC22A5 are the cause of systemic primary carnitine deficiency (CDSP), an autosomal recessive disorder manifested early in life by hypoketotic hypoglycemia and acute metabolic decompensation, and later in life by skeletal myopathy or cardiomyopathy.

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bio-techne®
Global bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL +1 612 379 2956
USA TEL 800 343 7475 Canada TEL 855 668 8722 China TEL +86 (21) 52380373
Europe | Middle East | Africa TEL +44 (0)1235 529449